high resolution. Strikingly, loss of TAT-5 disrupts cell contacts by causing the shedding of vesicles from the plasma membrane, likely explaining the morphogenetic defects we observed. The production of extracellular vesicles in TAT-5 embryos depends on the function of the recycling endosome-associated GTPase RAB-11, as well as the ESCRT complex, which normally acts in the formation of multivesicular bodies. Interestingly, these proteins have also been shown to regulate viral budding, a topologically similar process. Our findings define for the first time the essential role of a P4 ATPase in the regulation of PE asymmetry in a multicellular organism. Our results also suggest a novel mechanism whereby PE externalization influences dynamic remodeling of the plasma membrane during embryonic development.

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Program/Abstract # 361
A role for ADMP in scaling of embryonic tissues to generate equally proportioned embryos
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Vertebrate embryos originate from eggs of different sizes, which determine the size of the embryo during establishment, and patterning of the embryonic axes. Tissues and organs are scaled according to embryo size keeping their relative position in the body. We recently described a combined approach involving computational modeling and experimental manipulation to study the early BMP signaling pathway. By early/mid gastrula the BMP pathway establishes a signaling gradient that patterns the mesoderm along the dorsal–ventral axis. Our results showed that the main BMP ligand, BMP4, once bound to its antagonist, chordin, is efficiently shuttled to the ventral side to establish the activity gradient. Chordin expression localizes to the dorsal Spemann's organizer together with the BMP-type ligand ADMP, which can rescue embryos lacking BMP activity. The computational model predicted that ADMP is also important for the scaling of embryos along the dorsal–ventral axis. Analysis of ADMP and BMP4 expression along the dorsal–ventral axis in different sized embryos revealed higher ADMP transcript levels in larger embryos in agreement with the proposed model. Experimental manipulation of embryo size by dissection and explants showed, as expected, changes in ADMP expression levels. ADMP knock-down using an antisense approach, resulted in changes in gene expression. The results are consistent with scaling and in contradiction with ADMP directly contributing to the BMP gradient. These results suggest that ADMP performs multiple roles during early gastrulation. A model is proposed whereby a very early effect of ADMP regulates the scaling of the dorsal–ventral axis and subsequently participates in the BMP gradient to pattern the mesoderm.

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Program/Abstract # 362
Spatial patterning of muscle fibers in the *Xenopus laevis* embryo
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Mesoderm cells of the early embryo become the muscle fibers of the adult. During embryo development, mesoderm cells elongate into muscle fibers and form transient structures called somites. To understand how mesoderm cells of the early gastrula embryo organize to become the muscle fibers of the somites, we tracked cell movement in *Xenopus laevis* embryos. Using a cell-transplantation approach, we examined the movements that position these cells three dimensionally within somites and along the embryo's axis. We show that gastrula cells positioned in the upper lateral lip region (ULL) are directly adjacent to the notochord as they enter the presomitic mesoderm (PSM). These cells will eventually form muscle fibers in the central region of somites positioned along most of the anteroposterior axis. In contrast, cells positioned in the lower lip (LL) region of the gastrula migrate dorsally around the blastopore lip and appear to enter the PSM from the lateral edge. This population of cells then splits to flank ULL cells dorsally and ventrally to form muscle fibers in the dorsal and ventral quadrants of somites along the trunk and tail axis. Together, these results show that cells in the gastrula undergo different trajectories to give rise to muscle fibers positioned in distinct locations within somites and along the anteroposterior axis. These results offer new insights into how cells migrate as the embryo forms muscle, and contribute to our understanding of embryo development.

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Program/Abstract # 363
A genetic modifier screen identifies chromosomal intervals harboring potential midline interacting genes
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We are using a genetic modifier screen and RNA interference (RNAi) methodology to identify genes that interact with the Drosophila T-box transcription factor midline (mid). This screen assays a dosage-sensitive, eye-specific mutant phenotype observed when levels of midline are reduced in the developing eye imaginal disc by RNAi and placed within a second chromosomal deficiency background. Thus far, we have uncovered several third chromosomal intervals harboring potential mid-interacting genes. By chromosomal deficiency mapping, we have delimited the cytological intervals of interest within each deficiency line and are now obtaining mutant alleles of all candidate genes to determine which gene or genes interact with mid to: 1) affect sensory organ precursor formation giving rise to interommatidial bristles, 2) affect cell-fate specification in the embryonic CNS, 3) affect the formation of the ventral nerve cord via the regulation of axon guidance molecules and/or 4) affect other unique cell biological processes that are essential for proper eye and CNS development. The culmination of these studies will provide a greater understanding of mid function as a key component of conserved regulatory signaling pathways that guide the development of multiple tissues.

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Program/Abstract # 364
Stabilin2 is involved in Zebrafish arterial venous differentiation
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The hyaluronic receptor for endocytosis (Stabilin-2/HARE) mediates systemic clearance of multiple glycosaminoglycans including hyaluronan (HA), the chondroitin sulfates, heparin and others from the vascular and lymphatic circulations. In addition to its role in GAG clearance, recent in vitro studies indicate that Stab2 can participate in signal transduction by interacting with HA which results in ERK phosphorylation. However, it is